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THE RECOGNITION AND MANAGEMENT OF RHEUMATIC FEVER IN CHILDREN

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It is a real opportunity to appear before you today to speak on rheumatic fever, a disease which has become an important public health problem in our country both from the standpoint of morbidity and mortality. Consider these few figures—40,000 deaths a year with the average age of death 30 years. Besides this high mortality, rheumatic fever and rheumatic heart disease cause various degrees of physical incapacity in about 1% of the wage earning population. It is an important cause for rejection of soldiers and sailors in the armed forces, and it is expected that there will be an increase in the disease in over-crowded defense areas. Already there have been many cases in our military hospitals, especially amongst the naval personnel. In the school population it is estimated that there are about 1% of children with rheumatic heart disease, giving a total of about 200,000 so afflicted between the ages of 5 and 19. Considering all age groups, there are about 1,000,000 individuals afflicted. Mortality statistics show that rheumatic infection constitutes the major cause of death between 10 and 14 years of age, and runs a close second to tuberculosis between 15 and 24 years of age. These few figures give some idea of its importance as a public health problem.

Rheumatic fever is essentially a disease of childhood, the initial attack taking place usually between 5 and 15 years of age, with the average age of onset about 7 years. One of its chief characteristics is that it tends to recur following the initial infection, and this is the reason why it is so baffling and so unpredictable in its course. Fortunately, recurrences tend to diminish after puberty. In other words, the polycyclic types of rheumatic infection are more common before puberty and the monocyclic types after puberty. The highest mortality is in the first 5 years following the initial infection and it is therefore highly important that the frontal attack be made at this time when reactivations of the disease are so likely to occur.

In a group of 1438 children followed now for 22 years, 42% of the deaths (totaling 446) have taken place during these first 5 critical years. T. Duckett Jones has stated that of his deaths two-thirds have occurred in the first 5 years from the primary infection. It is not common for a child to die in the initial attack of rheumatic fever, but rather during a succession of recurrences. Most children who die succumb to their infection and not from congestive failure, although the latter may be superimposed. Congestive failure from rheumatic heart disease is more often encountered in adults.

In the approach to the control of rheumatic fever there are 3 cardinal principles to keep in mind:

1. Early recognition of the disease;
2. Early and prolonged treatment;
3. Prevention of recurrences.

In the early recognition and diagnosis of rheumatic fever, the burden of proof often falls on the physician. Many cases are extremely difficult to be certain about and may require months of observation. It is unnecessary to add here that accurate diagnosis is of vital import to the child and his parents and may affect his schooling and all his activities, which includes, in no small measure, his psyche. It is a humbling thought to quote Breed of Boston, in what has now almost become an aphorism, "We do not know when it starts, we often cannot recognize it when it is here, we have no cure for it and cannot tell when it is gone." But these words, written in 1932, sound worse than they are and strike too pessimistic a note. Much has been learned of the disease since that time, which gives us hope for the future.

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To the practicing physician and pediatrist, I wish to call attention to some of the minor patterns of rheumatic fever which may challenge us. A child about the time of the second dentition may be found by the parent or teacher at school to tire easily, to be losing weight, to have frequent nose-bleeds with increasing pallor and to complain of vague and fleeting pains in the extremities. Such a child may be found to have a slight fever, an increased heart rate, possibly an increase in the leucocyte count and probably an increased erythrocyte sedimentation rate. Other subacute and chronic infections of childhood may give similar symptoms, and our difficulty in making a diagnosis becomes apparent. At this early stage, the examination of the heart does not always help us, and although the heart is almost always involved in the rheumatic process, the valvulitis may not be clinically demonstrable until later in the course of the disease. However, the electrocardiogram may help us in the prolongation of the PR interval. Other more manifest symptoms and signs may make their appearance, such as typical multiple rheumatic joints (polyarthritis) with their extreme pain, the appearance of erythema multiforme, the finding of subcutaneous nodules, the presence of fever and tachycardia, and on examination of the heart, a gallop rhythm due to the appearance of a mid-diastolic sound, and the finding of a murmur usually systolic in time and heard best at the apex. At this stage, only time can evaluate the significance of such a murmur. All we can say is, that a carditis is probably present. It may be that a peri-cardial friction rub can be heard, which would strengthen the diagnosis of a rheumatic carditis.

The active phase of rheumatic fever may be ushered in by abdominal pain which may simulate appendicitis, and may test the acumen of the attending physician. This is a fairly frequent occurrence and has given us all concern. If in doubt, it is best to call in a good surgeon and have an exploratory laparotomy done. I have never seen such an operation do harm, and there should be no chagrin in making a wrong diagnosis. It is better to have the pathologist make a diagnosis for us in vivo, than to run the risk of over-looking an acute appendicitis with a spreading peritonitis and the death of the patient.

Chorea, which is a manifestation of the rheumatic state, may be over-looked by the physician when seen in the early stage. It is important to differentiate the irregular choreiform movements from nervous tics and habit spasms. Diagnosis is important because even mild types of chorea may be associated with severe rheumatic heart disease.

There is a common belief that rheumatism and polyarthritis are synonymous. This is not so, for the younger the child the less likely are the joints to be involved. The acutely swollen and painful joints are more often seen in the older child and young adult. I believe it is for this reason that rheumatic fever is so often missed in the very young. The presence of so-called "growing pains" have caused a misconception in the minds of many people. Their interpretation is not always easy, but it is important to differentiate between "growing pains" which are rheumatic and those which are non-rheumatic. Many children whom I have seen with carditis of severe degree have given no other history than that of "growing pains", with some of the minor patterns of the disease which I have described. Many rapidly growing children do complain of pains usually in the lower extremities at night. These are not rheumatic and in some cases can be attributed to flat feet or faulty posture. The differential diagnosis is important.

We have then, a disease which may be insidious in its onset, protean in its manifestations, polymorphic in its behavior patterns, unpredictable in its course and showing a tendency to be polycyclic. In a discussion of the management let us keep these facts in mind.

The early care of the child, that is, adequate and prolonged care following the first infection, should be stressed. In this regard, the treatment of rheumatic fever can be likened to the treatment of tuberculosis, in that it should be on a long term basis. This should be our objective and to remember that rheumatism, like the law, has a long arm. There is no reason why the child with rheumatic fever should not have as prolonged care as the child with tuberculosis.

Having instituted early and adequate care following the initial infection, the next important step in the management of the disease is the prevention of recurrences, which are more often the rule than

the exception. "Should I take my child to a tropical climate" is a question which many a parent poses to the doctor. No dogmatic answer can be given, and each case must be judged as an individual problem. The pros and the cons must be carefully weighed, for it is rarely possible to dislocate the life of an entire family, and the assurance of such a move to a southern clime cannot be stated categorically in terms of rheumatic recurrences. The question of climato-therapy, however, is rarely one to trouble the physician for the majority of our rheumatic children come from the lowest economic class and usually from homes of poverty and want. I am reminded of the adage, "If wishes were horses beggars could ride." Our problem and dilemma is not unlike that of the vitamin problem. Those who can afford them do not need them, and those who need them cannot afford them.

Barring a sojourn to the south, what then can we do to protect the rheumatic child? This becomes our main problem, and as physicians, nurses and public health workers, it is one to which we must direct ourselves. In a word, we should make every effort to protect the rheumatic child from upper respiratory infections caused by the Group A beta-hemolytic streptococcus. Such a statement is predicated upon our knowledge, almost universally accepted, that the hemolytic streptococcus, although not the cause of rheumatic fever, plays an important role in its genesis. The streptococcus may be likened to a detonator which sets off the unknown factor X resulting in rheumatic fever in the susceptible individual. It is well known that outbreaks of rheumatic fever have followed scarlet fever and streptococcal pharyngitis where large numbers of individuals are crowded together, as in barracks and boarding schools. This has been demonstrated in many of our military camps during the present war. A latent period of 7 to 21 days follows the streptococcal respiratory infection, which Coburn calls the pre-rheumatic phase and this is followed by the rheumatic phase in certain individuals susceptible to the disease. This is not unlike the invasion, latent phase and the onset of paralysis in polio-myelitis. In one child paralysis occurs, and another escapes. In one child rheumatic fever sets in, and another escapes. The immunity is largely dependent upon hereditary or acquired immune responses (or both) within the body mechanism. The child can best be protected by proper isolation in the hospital, a sanatarium or a convalescent home. If these are not possible, care can be given in a well supervised foster home. The latter is less costly and has certain advantages. In the setting up of State programs, such as you have done here in Rhode Island, I would commend to you this type of management for the rheumatic child.

In speaking of the streptococcus and its relation to the rheumatic state, a word should be mentioned concerning chemo-prophylaxis. The rationale in the use of the sulfonamides (especially sulfanilamide) has been based on the assumption that the prevention of streptococcus infection will prevent rheumatic recurrences. A number of studies have borne this out and have shown the effectiveness of this drug as a prophylactic agent. The decision to use chemo-therapy is a serious one for the drug has to be given over a period of many months. Severe toxic reactions have been encountered, and several fatalities have occurred. If sulfanilamide is used as a prophylactic, the physician should watch closely for these toxic reactions. I am sure that it is unnecessary to speak a word of caution against the use of the sulfonamides in the active phase of the rheumatic infection. They are definitely contra-indicated and usually do more harm than good. For prophylaxis they may be tried, but for therapy never, at least until our present state of knowledge is implemented by more factual clinical studies.

The word streptococcosis has been coined to express a chronic recurring infection caused by the streptococcus with a close relationship to the rheumatic state. An analogy to tuberculosis has been pointed out in the chronicity of the two diseases and their widespread dissemination throughout the body. The newer concept of rheumatic fever is that it is a systemic disease affecting many organs and tissues. The heart is the organ which, by its involvement, does affect the ultimate prognosis and the severity of the repetitive attacks of rheumatic fever, causing increasing cardiac damage, is the chief feature controlling the immediate outlook. Carditis, then, is the chief visceral manifestation of rheumatic fever and makes the disease the serious one it is.

As in tuberculosis, the best treatment for rheumatic carditis is complete rest in bed during the acute and sub-acute stage of the disease. Consider what bed-rest does for a child suffering from rheu-

matic heart disease, who has a tachycardia of say 120 beats per minute. If the heart can be slowed only 10 beats per minute by bed-rest, this means that in 24 hours it contracts nearly 15,000 less times. This, in itself, is a conserving process in a vital organ, which is so seriously affected by the rheumatic infection. Prolonged rest should therefore be continued until the active rheumatic infection has become quiescent.

An active educational program on rheumatic fever has been recently launched in this country. Your State is taking an active part, and has been forward looking in that the disease is now reportable. In such a campaign stress is put on early and exact diagnosis. As physicians we should exercise every care before we label a child as a cardiac. Too many children are being taken care of in heart clinics or by private physicians without justification. They have been condemned for years to a life of partial invalidism merely because of a heart murmur. This is indefensible. It is all too easy to develop an "invalid reaction" in these children. They grow up with feelings of insecurity, inferiority and anxiety states, and make up in adult life the group of so-called "cardiac neuroses". This, then, is the other side of the picture, and is included in this presentation merely as a plea for exact diagnosis. I would suggest that consultative cardiac clinics be set up, to which physicians can refer their patients for diagnosis, when they are in doubt or need the added aids which the X-ray, fluorescope, electro-cardiogram, sedimentation rate can give.

The care of the rheumatic cardiac child involves problems in education, in occupational therapy and in vocational guidance. Time does not permit to do more than mention these as an important part of the total program. We should always keep in mind that there is not only the heart to be treated, but there is the child who has the heart. We must not think of him as crippled but merely as handicapped. In his rehabilitation the public health nurse, the Social Service worker, the teacher and the occupational therapist play a large part.

In conclusion, I hope I have given you some conception of the size and importance of rheumatic fever as a public health problem. Rhode Island is one of 13 states which have set up State programs for the care of these children and young adults up to 21 years of age, and 5 other States have made application to the Children's Bureau. With the increasing dissemination of knowledge it is hoped that physicians, nurses, parents, teachers and all who have anything to do with children will have more of an awareness of rheumatic fever and will regard it as one of the most serious diseases of childhood.

EXTRAPERITONEAL CECOSTOMY

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The history of decompression of the obstructed colon has been one of slow and steady progress. Littrè in 1710 was the first to suggest and describe an intraperitoneal cecostomy. Pillore of Rouen, in 1776, performed this operation for the first time, through a McBurney incision and the opened cecum was sutured to the skin in this case. Duret in 1793 performed the first extraperitoneal colostomy. Callisen of Copenhagen in 1800 and then Amussat of Paris, championed and popularized extraperitoneal colostomy. The incision invariably was in the left lumbar region.

With the advent of asepsis and anesthesia, the intra-abdominal procedure of Littrè came back into prominence and the extraperitoneal technique faded into obscurity. Many modifications have been advocated, each a definite improvement on its predecessor. The indications for a cecostomy, a transverse colostomy or a sigmoidostomy have become definitely established. The necessity for a temporary or permanent artificial anus likewise is well understood.

A simple extraperitoneal cecostomy can be performed as follows: A McBurney, gridiron incision

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is made. It is wise to place the incision closer to the anterior-superior spine and slightly higher than the usual appendix incision. The external oblique fascia (and muscle, if one is that high) is cut obliquely. The internal oblique and the transversalis muscle are split transversely so as to expose the peritoneum. In the cases of acute obstruction of the left colon, with distention of the cecum, one finds that the reflection of the peritoneum over the cecum and onto the posterolateral abdominal wall, is elevated. That is, the peritoneal angle in the right lateral gutter, rises with the increasing distention of the cecum, thus increasing the area of unperitonealized cecum and ascending colon. This fact is taken advantage of in the next step of the operation.

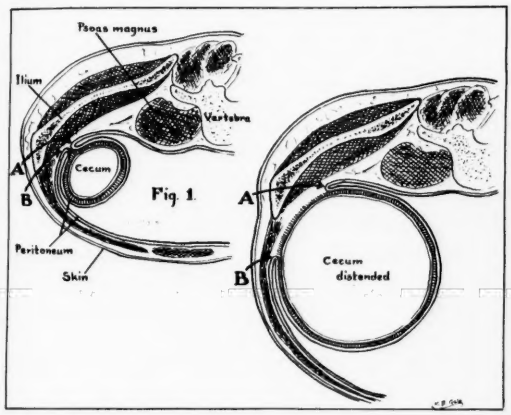

FIG. 1. Diagram demonstrates the increase in the unperitonealized portion of cecum present in cases with marked distention. See A-B. The reflection of the peritoneum at B can frequently be "elevated". Cecal wall can easily be brought up to skin level. See picture No. 2.

The peritoneum is pushed up and towards the midline so as to expose the posterior surface of the cecum or if need be, the ascending colon. This portion of the bowel is grasped with Babcock clamps, the bowel is further mobilized by finger dissection and can ordinarily be brought up to the skin level. Purse-string sutures are placed and the bowel is opened. A trochar is preferable, as it allows one to drain off the distended bowel without covering his field with feces. A larger catheter is then inserted and the purse-string suture is tied. A cigarette drain is inserted down to this region and sulfanilamide powder is sprinkled freely into the wound. The wound is then sutured loosely. If a small incision has been made, only one or two sutures are necessary. In our second case no sutures were used.

The placing of two Kocher clamps, as now practiced by Dr. Cattell, above and below the catheter so that each clamp catches about ¼ inch of the cecum, closest to the opening, is helpful, in that when the clamps are flattened down on the abdominal wall, they serve to prevent retraction of the cecum, when the bowel has been decompressed. They also destroy, by crushing, a little more of bowel wall, so that when the clamps and tube are removed after 72 hours, a good large opening in the cecum is obtained.

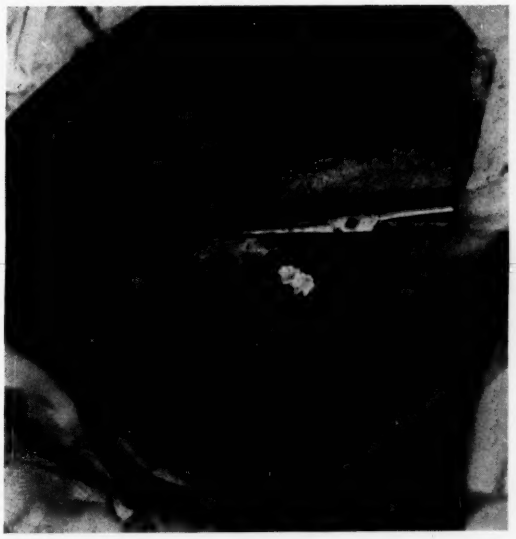

FIG. 2. Photograph of a patient. Note how clamps keep cecum on a level with skin. Drains can be seen below lower clamp. Note also size of incision and absence of sutures.

If one should discover at operation that the posterior surface of the cecum cannot be exposed because of the fixation of the lateral peritoneal reflection, then a two-stage transpertioneal procedure, such as has been advocated for drainage of appendiceal abscesses, can be used. That is, as soon as the peritoneum has been exposed, it is packed

against the lateral cecal wall with dry or vaseline gauze. After a suitable time interval, preferably 24 hours or more, the opening into the bowel can be made with a trochar and the catheter, etc., inserted.

In all cases of this type, a Miller-Abbott tube can be used in conjunction with the cecostomy. This is especially necessary in those cases in which a delayed type of operation is to be performed.

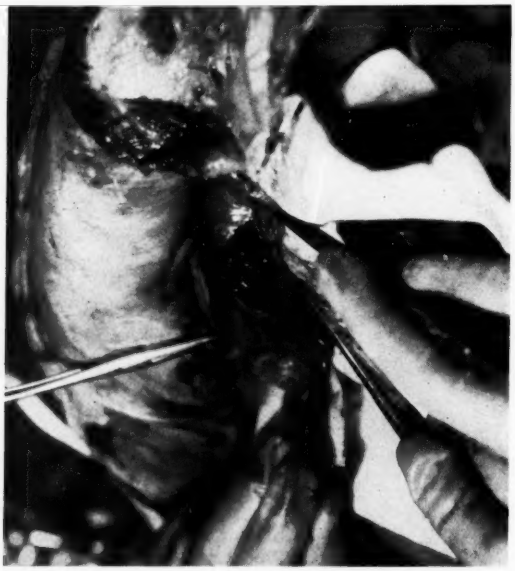

FIG. 3. Photograph at autopsy. Right anterior abdominal wall has been rolled out. The posterior peritoneum has been incised exposing retroperitoneal area. Thumb forceps hold one edge and clamp is placed on other edge. Cecostomy opening can be seen between the two margins of the incised postero-lateral peritoneal reflection.

Cecostomy, as a palliative operation preceding left colonic surgery, is often a life-saving measure. This is especially true in those first seen with marked obstruction. The intraperitoneal operation presents few technical difficulties, but a review of the results obtained in those cases that are first seen acutely obstructed and markedly distended, reveals an unusually high mortality rate, which is ordinarily attributed to the poor condition of the patient.

A critical review of the intraperitoneal procedure, however, would lead one to suspect that peritonitis, rather than the patient's condition, is the real offender. Contamination of the peritoneal cavity is inevitable, whether one uses a needle to deflate the distended cecum, purse-string sutures about a catheter, a trochar or a suction tube. Spillage about these, varies with the degree of bowel distention, the fluidity of the bowel contents, or the ease with which one introduces the catheter into the bowel lumen. Some leakage, no doubt, also occurs within the first 24 hours postoperatively, even though the catheter is brought through a hole in the omentum. Many will say that this contamination is insignificant, that the peritoneum is well able to take care of it or that the sulfa drugs will prevent peritonitis. These factors are a Godsend in the young, healthy and unobstructed patient but are they of as much help in a patient who has cancer, who has been obstructed for days, who is dehydrated and has a low serum protein as well as avitaminosis? This type of patient will not tolerate contamination of his peritoneal cavity and moreover will react unfavorably to anything except a minimal amount of handling.

Extraperitoneal cecostomy offers us an opportunity to decompress the bowel with no fear of peritonitis and with no possibility of handling or fighting the distended loops of small bowel, which invariably, when one uses a right rectus or McBurney incision, have to be walled off.

Some may fear an ascending infection in the retrocolic area and because of this, object to the extraperitoneal procedure. We have had no trouble with infection and feel that infection will usually take the path of least resistance. Thus if the region is packed with two to five grams of sulfanilamide or microcrystals of sulfathiazole and drained, the infection should remain well localized. Retrocecal appendiceal abscesses are often drained by this same procedure with no fear and rarely with any complications. It is considered as an ideal approach in these cases, by many surgeons.

Extraperitoneal cecostomy, because of this, is a safe procedure. Another distinct advantage is that we work in a virgin peritoneal cavity during the second stage. It is very simple and can be performed in much less time than the intraperitoneal operation. The operating time in our four cases has averaged twenty minutes, and all of our cases were carried out under local anesthesia.

It is wise preoperatively to take a flat plate of the abdomen in order, if possible, to locate the cecum. This will save embarrassment if one should encounter a high cecum.

The following four cases, each performed by a different surgeon, present many interesting facts. First let us consider the simplicity of the technique. The idea occurred to me while our surgical service was discussing the treatment of our first patient whom we believed would die if operated upon. Surprisingly, the operation was completed in twenty minutes and most of the time was spent collecting the feces, which exploded out of the cecostomy into a large basin.

The second case was operated upon by Dr. A. Eckstein, while I was standing by. He completed his operation in twenty minutes.

The third case was performed by Dr. O. Smith, who assisted me on the first patient. He encountered no difficulty.

The fourth patient was operated upon by Dr. E. Porter. I assisted at this operation and no complications developed. No case showed any signs of an ascending retrocolic infection.

In all these cases, the unperitonealized portion of the cecum was easily brought up to the skin margin and held there by clamps, even though several of the men had never seen the operation performed.

Three of these patients lived. The fourth one died twelve hours postoperatively. The cause of death as revealed by autopsy was a volvulus of an unusually large sigmoid with secondary thrombosis of the involved mesentery and gangrene of the bowel.

The three patients that lived were operated upon again. In all of these as well as in the patient that died, it was established that the peritoneal cavity was not entered into during the cecostomy operation, thus giving the surgeon the benefit of a virgin peritoneal cavity.

In my patient, I was able to resect a carcinoma of the midsigmoid and do an aseptic end-to-end anastomosis. This patient is living and well, seven months after his operation. The cecostomy closed spontaneously.

Dr. Porter's patient was found to have many metastatic lesions in the liver and glands and a carcinoma of the rectosigmoid. A loop sigmoidostomy was performed.

Dr. Eckstein's case is the only one in which the distention was not relieved. It was then realized that the obstruction was proximal to the cecum even though the original flat plates of the abdomen showed marked distention of the colon down to the sigmoid. At a second operation, exploration revealed metastatic lesions obstructing the small bowel. An ileostomy was performed. The progress was steadily downhill, and the patient died four weeks after admission.

These cases bring out the one fault of an extraperitoneal cecostomy. That is, its failure to allow an inspection of the abdomen and it is for that very reason that its use should be limited to those neglected cases that come into the hospital markedly distended and toxic. Here one cannot waste too much time with the Miller-Abbott tube and the usual cecostomy will not be tolerated. We feel that in these cases an extraperitoneal cecostomy is of distinct advantage.

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LEUCOPENIA IN INFECTIONS

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Leukopenia exists if the white cells in the circulating blood are definitely below their normal range of from five to ten thousand. Four thousand is the commonly accepted figure below which it is present.

We usually think that leukopenia is due to the decreased production of white cells, and this is no doubt the commonest and most important cause. There are, however, other possible mechanisms. Lawrence has divided them into five groups, the first of which is diminished production, and this group is again divided into four sub-groups. The first of these is a simple inhibition of the bone marrow, and this is the method by which leukopenia is commonly produced in infections. The second subgroup is the so-called maturation arrest, a process that occurs in at least some cases of agranulocytosis. The third is marked by aplasia of the bone marrow, as in aplastic anemia, and the fourth is due to infiltration of the bone marrow, as in tubercular disease affecting the bone marrow, or metastatic malignancy invading it, and crowding it enough to interfere with its function. Lawrence's second main group is increased leukocyte loss, as may occur with large purulent exudates in empyema, peritonitis or ulcerative colitis. The third is accelerated cell destruction in the circulating blood—this supposes the formation of a leukocytic toxin by infection or other pathological process. The fourth group is redistribution of the cells in the vascular channels, as occurs after the injection of a foreign protein, as typhoid vaccine, or after an allergic reaction. The cells are sequestrated in the internal organs—the liver, lungs, etc.—where they are commonly stored, and decrease in the peripheral blood. This is an interesting but temporary and unimportant process. The last group is that of redistribution of the cells in the body as a whole—the method in subleukemic leukemia, when there is leukopenia with infiltration of the body tissues with quantities of white cells.

As has been said, the main method of development of leukopenia in infection is the simple inhibition of the bone marrow. This is by some unknown toxin produced in certain infections. The mechanism may be simple or it may be covered with a cloak of simplicity that has as yet not been removed. The white cells are developed in the bone marrow in an orderly but retarded fashion. This process differs from that in some cases, at least, of agranulocytosis, where the development of the cells proceeds to the final stage of maturation but stops there. These cells, failing of complete maturation, are not released into the blood stream, and increase in the marrow. In infectious leukopenia, however, each step of development is carried out, but in a leisurely manner.

The infections which typically produce leukopenia are well known. There are certain bacteria, those of typhoid, the para-typhoid group, and tuberculosis. A few protozoal infections also are included, malaria being the only one of any consequence here, with Kala-azar and relapsing fever in the list. Virus infections that cause it are those of measles, German measles, and influenza. In addition there are other virus infections that are usually characterized by an initial leukopenia which is succeeded by a leukocytosis—these are smallpox, the virus pneumonitis which has been increasingly prevalent of late years, and infectious mononucleosis. In the hospital we usually find that our virus pneumonitis cases have a normal or slightly elevated white count—if the count is more than moderately elevated we are inclined to look for secondary or intercurrent infection or to question our diagnosis. Less than half of these cases show leukopenia. Probably if we had these cases at the onset and not after a week or so of their course, as we commonly do, we would have a higher incidence of leukopenia. As to mononucleosis, I think it is generally considered to be characterized by a moderate leukocytosis. The figures of a series of cases rather recently reported show, however, that a third of them never have a white count above ten thousand and that more than half have a definite leukopenia during the first week. In a condition which may have as varied manifestations as this one, this fact may well be borne in mind. Recently we had a case on the service with a high tempera-

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ture, no glandular enlargement and a leukopenia. Mononucleosis was not considered until it was suggested to us, in Saturday conference, by Dr. Fox. This diagnosis was confirmed—if we had been more aware of the frequency of the initial leukopenia we would probably not have missed it.

Leukopenia occasionally occurs in an infection in which leukocytosis is expected, and may have different implications. A case of pneumococcic pneumonia, say, has a low white count. His temperature is low, he is not toxic, and we assume that he has a mild infection which is being handled in stride. The bone marrow has not been markedly stimulated as there is no need to call out the reserves to handle the situation. We consider the prognosis to be good. A similar pneumonia with the same count is toxic, has high temperature, etc.—in his case we think that the bone marrow has not been stimulated but overwhelmed by a severe infection, and is unable to meet the call for increased white cells. We consider the prognosis in this case to be poor, especially if in addition to his low count there is an increase in the younger leukocytic forms—the nonfilamented ones—above their normal six per cent, and the smear shows the toxic granulizations and vacuolization of the cytoplasm that go with severe bone marrow effort.

A factor that may modify the leukocytic reaction is the location in the body of the infection. For instance, pulmonary tuberculosis commonly evokes a leukopenia, but in tubercular meningitis leukocytosis is the rule.

The inhibitory effect of infection on bone marrow is not usually an absolute one. If intercurrent pyogenic infections develop—as a bronchitis in the course of typhoid—there will be the usual leukocytosis, which will last until the bronchitis has cleared, and will then be replaced by leukopenia if the typhoid is still active. Zondek has recently reported similar white cell response, in typhoid, to certain bone marrow stimulating chemicals with which he has been working. In neither case has the typhoid be benefited by the leukocytosis.

THE AMERICAN CONGRESS OF PHYSICAL THERAPY

Will hold its twenty-second annual scientific and clinical session September 8, 9, 10 and 11, 1943, inclusive, at the Palmer House, Chicago. Rehabilitation is in the spotlight today—Physical Therapy plays an important part in this work. The annual instruction course will be held from 8:00 to 10:30 A.M., and from 1:00 to 2:00 P.M. during the days of September 8, 9 and 10, and will include a round table discussion group from 9:00 to 10:30 A.M., Thursday, September 9. The scientific and clinical sessions will be given on the remaining portions of these days and evenings. A feature will be an hour demonstration showing technic from 5:00 to 6:00 P.M. during the days of September 8, 9 and 10. All of these sessions will be open to the members of the regular medical profession and their qualified aids. For information concerning the instruction course and program of the convention proper, address the American Congress of Physical Therapy, 30 North Michigan Avenue, Chicago, Illinois.

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106 Francis Street, Providence, R. I.

DR. CHARLES F. GORMLY

On June 26th Dr. Charles F. Gormly died. The factual story of his career is given in the address by Dr. Hammond, published in the June issue of this JOURNAL. The value of his life to his friends and colleagues, to his casual acquaintances, indeed to every citizen of Rhode Island, is beyond our ability to estimate.

Throughout the thirty-one years of his active practice he has always been a dynamic force for the betterment of the condition of his patients, his medical associates, his hospital and his community. Up to the very time of his death he preserved a cheerful optimism and a clear-sighted interest in the planning of a future in which he knew he could not share.

Always, even in the face of inevitable physical suffering and disaster his ready wit and glowing humor never failed. For almost a year and a half, as was generally known, he suffered the progressive inroads of an incurable malady and yet he carried on undaunted as practitioner, consultant and physician-in-chief and in addition brought to a most successful conclusion his year as President of the Rhode Island Medical Society.

The courage with which he fought this campaign through equals the most heroic deeds for which men are decorated on the field of battle. He is a life-long inspiration to us all.

REORGANIZATION

With the next number of this JOURNAL Mr. John E. Farrell, now Executive Secretary of the Rhode Island Medical Society, will take over his duties as Managing Editor. His brilliant brain child, the *Providence Medical News,* will cease to exist in its present being, but by a happy metempsychosis will continue to flourish in our columns.

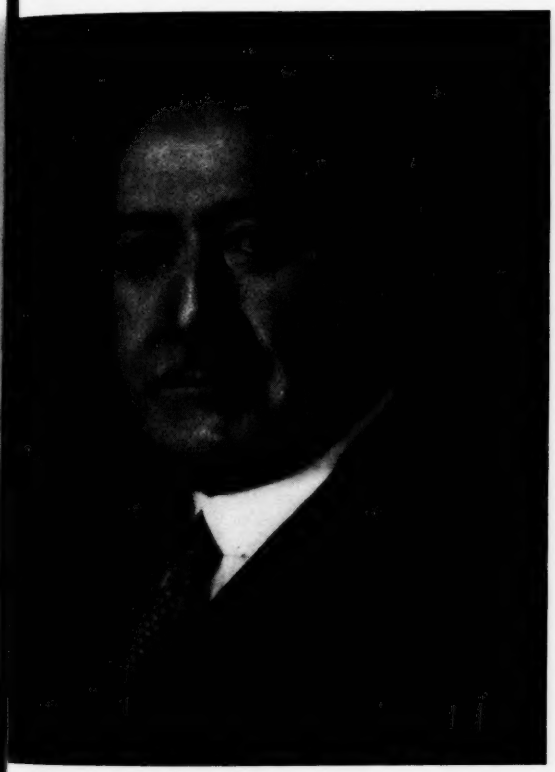
It was unique in its concept and development and has been flattered by numerous imitators. All the interesting and important subject matters of news, economic and sociological value that Mr. Farrell has so brilliantly found, recognized and presented will appear in a new setting. As a part of our old and honored Medical Society it should have even wider fields for progress.

The Society has grown not only in membership during late years but has shown a new liveliness and power. The local Association with its frequent meetings had, even before the reorganization of a few years ago, developed an initiative in supplying the needs of a major portion of the profession in the state. This had reacted unfavorably on the parent group, which is now coming into its own again. It is going to need a better journal with a broader scope. The teamwork will undoubtedly be mutually helpful.

The News was a development of the notices of meetings by the Providence Association. The JOURNAL will take over this duty and therefore will be published in the last week of each month. At present, due to war conditions, (the facile explanation of any failure to function properly) the JOURNAL is behind its publication schedule. This will be made up during the summer, and in the fall when meetings are renewed, it will perforce appear at the proper time.

The present editorial board is composed largely of members in the armed forces. Hence there will be a readjustment. To those who have served us in the past our thanks are given and when better times arrive we may ask for their help again.

With Mr. Farrell's great business ability, energy and flair for correlating our diverse interests we confidently predict a pleasing future.



PRESIDENT MICHAEL H. SULLIVAN

We congratulate Dr. Michael H. Sullivan on his accession to the Presidency of the Rhode Island Medical Society.

It is fifteen years since Newport was thus represented by Dr. Norman MacLeod, who filled the office so efficiently in those happy days of long ago.

Two centuries or so back Newport was the great seaport of the country, and Providence just an up the bay place that some of the smaller craft could reach. Then, as a number of historical articles in the JOURNAL have made clear, the same proportion held in medical matters. But manufactures and commerce have made Providence big and the overwhelming number of physicians have congregated here. Hence it is only rarely we now get our leader from the Island of Aquidneck.

Dr. Sullivan is the dynamic type to which we are accustomed in our Presidency. Almost at the beginning of his career he had a tremendous obstetrical practice in addition to his other work. He has served on the Board of Health over twenty years and is now vice-president; in both wars he has been active in the draft work.

He has been president of the Newport Medical Society and the staff of the Newport Hospital. With many years on the surgical staff he became chief of the obstetrical service when that was organized.

With all this experience he is well equipped to lead our organization and we predict a good and interesting year under him.

NICHOLAUS COPERNICUS, PHYSICIAN

Four hundred years ago, in 1543, appeared two immortal books. In May of that year an old man, about to die, held before his dimming eyes the first copy of *De Revolutionibus Orbium Coelestium*. He was Nicholaus Copernicus. In June of that same year a young man gave to the world *De Fabrica Humani Corporis*. He was Andreas Vesalius. Thus were born within one month of each other modern astronomy and modern anatomy, both of them the offspring of physicians.

Nicholaus Copernicus was born in Poland on February 19, 1473, and spent his childhood in his native town. When his father died he was placed under the guardianship of his maternal uncle, a man of distinction who was later a bishop and Polish Senator. Under the tutelage of his uncle the young Copernicus prepared himself for the pursuit of higher studies and from 1491 to 1495 he was a student at the ancient Polish University of Krakow which for the first time in 575 years has been closed by the Nazi barbarians. While studying at the University two things happened to Copernicus which influenced greatly his future interests and studies.

He came under the spell of the brilliant humanist astronomer and mathematician Albert Bruzewski, who was undoubtedly the first to arouse in his young student a love for the ancient science of astronomy. The second determining event in Copernicus' intellectual life was the arrival at the University of four astronomical instruments. As Dr. Stephen C. Mizwa informs us these were brought from Buda, Hungary as gifts to the University of Krakow. There was a large celestial globe, two beautiful astrolabes and a triquetrum. The arrival of these instruments created such a sensation that the Rector called a special student assembly to behold and admire these wonders. They have been preserved by the University to this day, and we may be sure that Copernicus was one of the most interested spectators.

For further study Copernicus journeyed to Italy which then was for earnest students sancta mater studiorum. At Bologna he enrolled as a student of canon law which in those days was essential to the training of a prospective Churchman. In 1500 we find him in Rome lecturing on mathematics and astronomy. From 1501 to 1503 he studied medicine at the University of Padua and at the same time obtained a doctor's degree in canon law at Ferrara.

Enriched with multifarious learning he returned to Poland and entered upon his duties as canon of the duchy-bishopric of Varmia. He was occupied with many things, acting as physician and secretary to his uncle Bishop Lucas, devoting much time to the healing of the sick-poor, continuing his astronomical observations and taking an active part in the politics of the day. It was in Varmia that Copernicus pursued his astronomical studies and tabulated the immense collection of data which culminated in *De Revolutionibus,* and it was there that he died on May 24, 1543.

To-day the ancient Polish people are being exterminated by the sub-bestial savagery of the German invader just as their forebears were harassed by the Teutonic Knights in the lifetime of Copernicus. But in spite of the darkness which covers their land, Copernicus still stands before his desolate countrymen as a star of hope and an incentive to endure. When the Germans entered Poland in 1939 they announced that they would make a "cultural wilderness" out of the land they conquered. They have done their best to make good their promise before a shocked and outraged world. And although the memory of Copernicus may not be publicly honored in his native land, it is heartening to know that here in America the Copernican Quadricentennial National Committee composed of distinguished scientists, scholars and educators has been devoting itself to the furtherance of commemorative exercises in honor of the great astronomer. And we physicians should be, as indeed we are, pleased to join in this expression of esteem for one of our profession, since Copernicus is worthy of remembrance not as a great astronomer only but also as a humble physician, a medical alumnus of "fair Padua", the prolific mother of Linacre, Vesalius and Harvey.

JOHN E. DONLEY, M.D.

RHODE ISLAND MEDICAL SOCIETY

REPORT OF THE COMMITTEE ON EDUCATION: STATE AND NATIONAL

Herewith is submitted the report of the Committee on Education of the Rhode Island Medical Society for the year commencing June 1, 1942.

The fifteen-minute radio talks which have now been running continuously since December 1938, over Station WPRO every Sunday afternoon at 1:30 P. M., have been continued without letup, and it is apparent from the many letters and requests for copies of talks that our audience is a growing and enthusiastic one. Forty-six radio talks have been given in the past year.

1,015 letters have been received by your committee this year from the general public requesting copies of these talks, and this same number have been mimeographed and mailed to the radio audience. There are six who call for a copy of each lecture. This regular mailing list includes Miss MacDonald at the Public Health Library, Miss Carberry of the Rhode Island State Department of Social Welfare and Mr. Dunham from the Red Cross. Many of the letters received contain not only requests but expressions of appreciation.

It is the belief of your committee that these radio talks should again be continued as in the past. We wish to express our thanks to Miss Dickerman and her associates and to Mr. John Farrell, Executive Secretary of the Providence District Society, for the work and assistance which they have rendered to the committee. We wish also to express our thanks to Station WPRO for its courtesy in giving us time, to Blanding & Blanding, Inc., for the advertising which has been freely contributed, and to the many members of the profession who have by their cooperation and suggestions been of invaluable aid.

Respectfully submitted,
JESSE P. EDDY, 3RD., M.D., *Chairman*

COMMITTEE ON NECROLOGY

Your Committee on Necrology herewith submits its report of deaths of members of this Society which have occurred during the past year:

Frederick N. Brown, M.D., Providence. Died May 23, 1942.

F. Edward Burke, M.D., Wakefield. Died October 21, 1942.

John P. Cooney, M.D., Providence. Died January 15, 1943.

Preston D. Geiger, M.D., Providence. Died October 24, 1942.

Henry W. Hopkins, M.D., Warren. Died October 14, 1942.

Patrick H. Keefe, M.D., Providence. Died June 4, 1942.

Thomas F. Kennedy, M.D., Woonsocket. Died March 10, 1943.

Captain Thomas A. Martin, MC, AUS, Providence. Died August 4, 1942.

Ardashes H. Merdinyan, M.D., Pawtucket. Died November 26, 1942.

Charles B. O'Rourke, M.D., East Providence. Died February 13, 1943.

Charles N. Raymond, M.D., Providence. Died September 19, 1942.

Charles W. Stewart, M.D., Newport. Died March 26, 1943.

Charles A. Sylvia, M.D., Providence. Died November 12, 1942.

Respectfully submitted,
EDWARD T. STREKER, M.D., *Chairman*

REPORT OF THE COMMITTEE ON THE LIBRARY

Because of prevailing conditions the activities of the Library have somewhat changed during the past year. Many of the Fellows who were among the most regular users of the books and periodicals have left for service in the various branches of the armed forces. On the other hand, the Library has been consulted constantly by officers who are stationed in this state and in Connecticut. Early last year it was deemed proper and fitting to extend the privileges of the Library, with certain restrictions, to all such officers, and the result has been that the Library personnel has been able to answer many inquiries, and to do much reference work by telephone.

The total number of visitors at the Library has been less than for some years, but there has been a corresponding increase in the number of consultations by telephone.

Statistics for the year:

Number of visitors	1,411
Journals bound	95
Journals repaired	4
Books received, through purchase, gifts and from the R. I. MEDICAL JOURNAL, sent for review	96
Journals received, by subscription, and exchange for the R. I. MEDICAL JOURNAL	132
Number of books in the Library, April 18, 1943	33,168
Books and Journals catalogued to April 18, 1943	21,073

Gifts from Fellows:

Dr. H. H. Armington	15 volumes
Dr. R. S. Wilcox	5 volumes

Journals from Drs. J. E. Mowry, R. Hammond, F. V. Corrigan and L. I. Kramer.

Circulation: 335 Journals and 120 books
Interlibrary loan requests, 9.

Respectfully submitted,
HERBERT G. PARTRIDGE, *Chairman.*

REPORT OF THE CHAIRMAN OF THE BOARD OF TRUSTEES OF THE RHODE ISLAND MEDICAL SOCIETY BUILDING

April 17, 1942 — April 22, 1943

Number of meetings held in the building, 48; inclusive of meetings of the Providence Medical Association. Also, two outside organizations held meetings in the R. I. Medical Society Building. This is the smallest number of meetings for which we have received donations in any one year.

The Rhode Island Recruiting Board of the Procurement and Assignment Division of the U. S. Army was granted the temporary use of the building from May 1942 to October 21, 1942.

The outside of the building has been painted and all the screens have been repaired and painted apart from other minor repairs.

Respectfully submitted,

ELIHU S. WING, *Chairman*

REPORT OF THE TRUSTEES OF THE FISKE FUND

At the present time, the moneys left by Dr. Caleb Fiske to encourage original work on the part of the members of this Society, amount to a little over $12,000. These funds are invested by court order in such a way that the income, under present conditions, is almost negligible; in fact, it is so small that the Trustees have felt unable to offer any premium this year, or to carry on any of the normal activities of the Fund. They are making efforts, however, to have the investments changed in such a way as to bring about a reasonable return, and they hope to be able to continue the work of the Fund next year.

Respectfully submitted for the Trustees,

WILFRED PICKLES, M.D., *Secretary*

BOOK REVIEWS

A MANUAL OF CLINICAL THERAPEUTICS (A Guide for Students and Practitioners), xv 609 pp. By Windsor C. Cutting, M.D., Associate Professor of Therapeutics, Stanford University School of Medicine. W. B. Saunders Company, 1943.

To encompass the entire field of therapeutic knowledge into one brief volume is a vast undertaking, yet that task is attempted in this fairly small and short book. The material is organized in the usual fashion — infections and infestations, virus diseases, endocrine and metabolic disturbances, allergy, physical phenomena, and finally groups of disease entities associated with various body systems. To this text is added in appendiceal form data on therapeutic procedures, physiotherapy, poisonings, diatetics, prescription writing and drugs, normal values, and common laboratory methods for controlling and estimating dosage.

When the author chose the name clinical therapeutics, he did not mean that this was to be a work on pharmacology, but to include general advice, physiotherapy, and psychotherapy. Indeed the scope of the book is so broad that there is a rather sketchy treatment of many subjects, particularly in the management of tropical and verminal infections and infestations, and diseases associated with the gastro-intestinal tract. Likewise the work suffers from the inclusion of rather rare and fancy conditions in occasional instances, many of these of a surgical nature, e. g. thrombosis of the superior mesenteric artery.

On the positive side of the ledger there is an admirable restraint exercised by Dr. Cutting in the choice of drugs and pharmaceutical agents, and as one reads there approaches a feeling of confidence that results can be expected where they are promised. There are several simple prescriptions included, and values are given in both metric and Latin systems. The author feels that the metric system should be universally adopted, and uses simple English for his directions. The recommendations for the use of sulfonamides are very circumspect. References to the periodical literature which is usually readily available and which appear after each small section would appear to be a thoughtful and helpful feature. On the whole the compilations of data in the appendices are more useful than the text itself, and add greatly to the value of the book.

HAROLD S. BARRETT, M.D.

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